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Veröffentlichungsversion / Published Version
Zeitschriftenartikel / journal article

Empfohlene Zitierung / Suggested Citation:

Floyd, K. (2014). Humans are people, too: nurturing an appreciation for nature in communication research. *Review of Communication Research*, 2, 1-29. <https://doi.org/10.12840/issn.2255-4165.2014.02.01.001>

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Humans Are People, Too: Nurturing an Appreciation for Nature in Communication Research

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Highlights

- Human social behavior, including communication behavior, is influenced by higher-order factors that remain largely unacknowledged in communication theory and research.
- Several communicative behaviors, including communication anxiety, conflict, emotional expression, and aggression, have strong biological ties.
- Most of the research on the biology of communication behavior is conducted outside of the communication discipline and remains unknown to communication scholars, even though the focus is on communication behavior.
- Ignorance of biology leads communication theory to be needlessly anthropocentric, offering human-specific explanations for behaviors that are not unique to humans.
- Communication research often ignores heritability and treats entities such as media and culture as though they were living organisms, when in fact they are human creations.
- Environmental influences such as enculturation, modeling, and media messages are powerful, but all environmental influences require biological factors to be effective.

Abstract

A growing literature illuminates the biological and evolutionary antecedents, consequences, and correlates of communication behavior. With few exceptions, however, the research is conducted outside of the communication discipline and remains unknown within the communication field. This essay offers illustrative literature reviews for several communicative behaviors and argues that the communication discipline should embrace, rather than ignore, the evolutionary factors involved in human social behavior.

Suggested citation: Floyd, K. (2014). Humans Are People, Too: Nurturing an Appreciation for Nature in Communication Research. *Review of Communication Research*, 2(1), 1-29. doi: 10.12840/issn.2255-4165.2014.02.01.001

Keywords: Biology, Evolution, Social Behavior, Neuroscience, Immunology

Editor: Giorgio P. De Marchis, Universidad Complutense de Madrid, Madrid, Spain

Received: Feb. 6th, 2013 **Open peer review:** July 15th **Accepted:** Oct. 15th **Prepublished online:** Nov. 11th
Published: Jan. 2014

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Who knows what I want to do? Who knows what anyone wants to do? How can you be sure about something like that? Isn't it all a question of brain chemistry, signals going back and forth, electrical energy in the cortex? How do you know whether something is really what you want to do or just some kind of nerve impulse in the brain? Some minor little activity takes place somewhere in this unimportant place in one of the brain hemispheres and suddenly I want to go to Montana or I don't want to go to Montana.

— Don DeLillo, *White Noise*

Children are perhaps the only beings to outpace professional scholars in their inquisitiveness. *Why?*—a question we ask about communication—is routinely offered by youngsters about a wide range of behaviors of which they attempt to make sense. We can, of course, answer a *why* question at multiple levels of abstraction. Why, for instance, do I get hungry each night around 6 pm? There are proximal environmental causes for my hunger: because I have not eaten since noon; because I have been conditioned by cultural or familial norms to eat every evening at that time. Proximal causes are so named because they are proximal to our conscious experience and therefore easy to articulate, understand, and even modify. But there are other reasons why I get hungry: because my level of the peptide hormone ghrelin is elevated; because I will perish without regular nourishment. Those seem further removed from our conscious experience, so they are easier to disregard, either because we are unaware of their occurrence (as we may be of ghrelin elevation) or because they are outside of our willful control (as is the adaptive need for regular nourishment).

Like gastronomic questions, communication questions can also be answered at multiple levels of abstraction. Although they can be, however, they often are not. Rather, communication theory and research practice (writ large) point fluently toward cultural, environmental, cognitively acquired causes of behavior while barely glancing at—if acknowledging at all—the biological processes or evolved adaptations that may also be influential. It is as if we fully recognize that we are cultural, political, and symbolic beings inhabiting a socially constructed universe but forget that we are also biological beings inhabiting a

physical universe. To the extent this is true, it is to our profound intellectual detriment. The human communication experience is older than culture and politics—older even than language—so to appreciate it, we must learn to think beyond ourselves and our proximal environmental conditions. Knowing the human social animal requires understanding *both* the social *and* the animal in the human.

As relevant as communication is, the communication discipline risks intellectual stagnation and academic irrelevance by continuing to underappreciate the influence of biology on human social behavior. To support that claim, the essay begins by defining and clarifying the problem of the discipline's lack of attention to biological influences. Next, a review is offered of several areas where fruitful research has been accomplished to demonstrate anatomical and physiological connections to communication behavior. Third, this essay explains how ignoring biological influences has led the field to some tenuous theoretic positions. Finally, a suggestion is offered for embracing the contributions of both nature and nurture as causal factors in human social behavior.

Defining the Problem:

Life Before *Homo narrans*

In contradistinction to proximal causes for behavior are those known to biologists as ultimate causes. Ultimate causation explains behavior with reference to the evolutionary forces acting upon it. The ultimate cause of an individual's behavioral tendency is the function that behavioral tendency serves with respect to that individual's survival and/or the reproduction of that individual's genes. Proximal and ultimate causes are not necessarily competing explanations for behavior. For instance, the tendency to act aggressively when threatened may have multiple proximal causes, including parental and peer modeling and reinforcement, exposure to media violence, gender role ideology, and cultural norms, yet also have the ultimate cause of advancing the organism's survival.

A consequential difference between proximal and ultimate causes, however, is that ultimate causes have shaped the evolution of the organism itself, through the forces of natural selection and sexual selection. Ultimate causes, therefore, influence the organism via innate path-

ways. No individual must *learn* to elevate ghrelin on a routine basis so as to induce the hunger pangs that motivate regular eating. That connection is presented as a part of normal human development. Proximal causes, however, are influential only through acquired pathways. No cultural or familial tradition, for example, can have any effect on an individual's eating behavior until that tradition is learned.

This difference in causal systems has some acute implications for the study of human communication. Humans—that is, those of the *Homo* genus—were not born into a world of language and culture, economics and religion, gender and psychology, Instagram and Twitter. Despite Fisher's (1987) endearing metaphor, the human species is not, in fact, best understood as *Homo narrans*—those who tell stories. It is, instead, best understood relative to its place in the natural world: as a social primate.

Who We Are

Humans are mammals, primates of the family *Hominidae*. The earliest species of human, *Homo habilis*, appears to have evolved around 2.3 million years ago, whereas *Homo sapiens* are thought to have evolved approximately 200,000 years ago (McDougall, Brown, & Fleagle, 2005). Agriculture is 10,000 to 13,000 years old at most (Hancock, 2012), meaning that for 90% of its history, *H. sapiens* lived as hunter-gatherers. Considerable disagreement exists among scholars as to whether language evolved slowly over the history of human evolution or appeared suddenly, perhaps due to a genetic mutation, as early as 50,000 years ago (see, e.g., Nichols, 1998; Perreault & Mathew, 2012). Despite the specifics, much of human evolutionary history has not been characterized by the experience of language—let alone the industrial, technological, cultural, financial, or political experiences that typify the modern human species.

It would be profoundly naïve to believe that modern developments erase the effects of thousands of years of evolutionary pressures on human behavior. To assume, for instance, that modern humans interact with each other in ways that transcend the vast majority of their evolutionary development—ways that entirely belie their human nature, that is—would be intellectually vacuous. A perusal of theory and pedagogy in the communication discipline, however, reveals a distressing ignorance of the

human as an evolved biological being.

Biology in the Communication Field

Given that the communication discipline focuses all of its academic attention on the behavior of one species—the human—it seems reasonable to assume that theory and pedagogy in the field would include consideration of anatomy, physiology, and evolutionary biology as antecedents to, consequences of, or correlates of human behavior, alongside other categories of causes, outcomes, and correlates. This is standard practice in allied social sciences. Introductory undergraduate textbooks in psychology (Kalat, 2013), family studies (Bernardes, 1997), and anthropology (Jurmain, Kilgore, Trevathan, & Ciochon, 2012) address biological and evolutionary influences on behavior directly in their foundational chapters. The proposition that social behavior affects and is affected by biological characteristics, some of which have been shaped by evolutionary pressures, is largely uncontroversial in the social sciences, writ large.

As Sherry (2004), Floyd (Floyd & Afifi, 2012; Floyd & Cole, 2009; Floyd & Haynes, 2005), and Beatty and McCroskey (Beatty, McCroskey, & Pence, 2009; Beatty, McCroskey, & Valencic, 2001) have noted, the reaction of the communication discipline to bio-evolutionary explanations for behavior has ranged from apathy to antagonism. Sherry correctly notes that of the seven principal theoretic traditions articulated by Craig (1999) in his seminal article, not one embraces “an ontology that acknowledges any contribution of biology in determining human communication behavior” (Sherry, 2004, p. 91). Even communication theories that posit a role for physiological arousal, such as Cappella and Greene's (1982) discrepancy arousal theory and Tannenbaum and Zillmann's (1975) arousal model, acknowledge arousal only as an outcome of proximal environmental influences such as parental behavior or media content, not as something with innate influences. Until recently, no communication theories have directly posited either biological or evolutionary causes for communication behavior or physiological, health-related outcomes of communication behavior.

That is not to say that research examining the links between communication behavior and biology is not being conducted. It is—for a number of communication behaviors and biological outcomes—but most often by

researchers outside of the communication field. This literature, although fairly voluminous, remains largely unknown within the communication discipline. The vast majority of it is unrepresented in communication textbooks, handbooks, anthologies, and other repositories of disciplinary knowledge. This suggests that the communication discipline is either ignorant of or actively ignoring what other fields are discovering about communication behaviors.

Communication and Biology: A Look at What We Know

It will come as a surprise to some communication scholars that the biological factors involved in communication behavior are actively studied. That is because, with few exceptions, they are actively studied outside of the communication discipline. An exhaustive review of published research linking communication behaviors to biological factors would be beyond the scope of this article. In its place appears a representative review focused on six diverse behaviors: communication apprehension/public speaking anxiety; affectionate communication; conflict; social support; emotional communication; and, aggression. Each behavior is a frequent focus of study within the communication discipline, yet with the exception of affectionate communication, the research identifying biological and physiological links to the behavior has been conducted principally outside of the communication field.

For each behavior, this review explains from the bioevolutionary perspective why the behavior might have been shaped by evolutionary pressures to have extant physiological substrates. This is an important, often absent, step when investigating the physiological dimensions of a social behavior. The review then offers a representative look at research connecting the behavior to neurological, cardiovascular, endocrine, immunological, hematological, and/or genetic factors.

Communication Apprehension/Public Speaking Anxiety

One of the first communication behaviors to be studied from a bioevolutionary perspective was communication apprehension (CA), also referred to as public speak-

ing anxiety, which may seem at first glance to be an unwieldy candidate for such an approach. Given that the history of the species predates the evolution of language itself, surely a fear of public speaking did not enhance the survival and reproductive prospects of humans' ancient ancestors. How it therefore could be subjected to the pressures of natural selection would remain inexplicable if not for the observation that it is not the fear of public speaking per se—but rather, the fear of social exclusion—that is adaptive.

CA—or its more colloquial term *stage fright*—is induced not only by public speaking but also by any performative opportunity that contains the risk of embarrassment. As a form of physical and emotional stress, embarrassment is highly adaptive insofar as it motivates humans to acknowledge and make amends for social transgressions, diminishing the likelihood of rejection or exclusion from one's social unit (see Miller & Leary, 1992). For a social species such as *Homo sapiens*, inclusion is critical to survival. In premodernity, being shunned from one's social unit would have meant a loss of access to communal resources such as food, shelter, and protection, dramatically reducing the chances for survival. Even among modern humans, social exclusion predicts physical pain (Macdonald & Leary, 2005), illicit drug use (March, Ovideo-Joekes, & Romero, 2006), and risk of suicide (Van Orden, Witte, Gordon, Bender, & Joiner, 2008). The bioevolutionary claim is not that public speaking anxiety leads to drug abuse and suicide. It is that a fear of social exclusion has been favored by natural selection, and that CA/public speaking anxiety represents one manifestation of that adaptation.

Public speaking is a stressor.

If true, public speaking should be not only commonly and widely feared—as, in fact, it is for both adults (Furmark et al., 2002) and adolescents (Essau, Conradt, & Petermann, 1999)—but also accompanied by an innate (i.e., unlearned) stress reaction. A robust research literature supports the latter prediction. Even irrespective of individual levels of CA, the task of presenting a speech elevates multiple physiological stress markers, including heart rate (Beatty & Behnke, 1991; Behnke & Sawyer, 2001), blood pressure (Lacy et al., 1995), adrenocorticotrophic hormone (ACTH) (Kirschbaum, Pirke, & Hellham-

mer, 1993), cortisol (Roberts, Sawyer, & Behnke, 2004), interleukin-6, a proinflammatory chemical related to stress (von Känel, Kudielka, Preckel, Hanebuth, & Fischer, 2006), soluble receptor for tumor necrosis factor- α (sTNF α RII), involved in the inflammatory stress response (Slavich, Way, Eisenberger, & Taylor, 2010), adrenaline and norepinephrine (Levine et al., 1985), and the circulating immune system cells CD3+, CD16+, and CD56+ (Lucas et al., 2006). Even among children and adolescents, public speaking elevates both cortisol and the stress enzyme α -amylase, an effect that grows stronger with pubertal development (van den Bos, de Rooij, Miers, Bokhorst, & Westerberg, in press). Notably, these markers reflect a cross-section of physiological systems, including the central nervous system, cardiovascular system, endocrine system, and immune system.

Public speaking is, in fact, so anxiety provoking that even the anticipation of a speech induces a stress reaction. Whereas Behnke and Sawyer (1999) have well documented this effect with respect to psychological stress and anxiety, it occurs for physiological stress as well. Anticipating a speech elevates heart rate (Davidson, Marshall, Tomarken, & Henriques, 2000; Preston, Buchanan, Stansfield, & Bechara, 2007), blood pressure (Lepore, Allen, & Evans, 1993), and cortisol and α -amylase (Starcke, Wolf, Markowitsch, & Brand, 2008). Lepore et al. (1993) found that blood pressure elevations are moderated by the presence of social support, however, and Gonzalez-Bono et al. (2002) found that anticipatory increases in heart rate are moderated by cognitive anxiety, at least for women.

Stress responses to public speaking vary individually.

As the CA research makes clear, however, public speaking is not equally stress inducing for everyone. Like many adaptive characteristics, CA shows individual variation, and some researchers have speculated as to the source of that variation. For instance, Beatty, McCroskey, and Heisel (1998) conceptualized CA with reference to Gray's (1970) biopsychological theory of personality, which posits the existence of two neuropsychological systems that control behavioral activity. According to Gray, the *behavioral inhibition system* (BIS) regulates an individual's reaction to anxiety-relevant cues in the environment (such as scary or sad events) whereas the *behavioral activation system*

(BAS) regulates reaction to reward-relevant cues (such as exciting events or attractive people). Beatty and colleagues (1998) argued that high CA results from an over-active BIS; that is, people with high communication apprehension seek to avoid public speaking situations because they become too physiologically aroused.

A logical extension of that argument is the prediction that stress reactions to public speaking are moderated by individual levels of CA or public speaking anxiety, and existing data support that claim. For instance, Roberts et al. (2004) found that cortisol levels during college students' speeches were linearly related to their self-reported speech anxiety scores. Other research has found that high-CA communicators are more likely than their low-CA counterparts to experience cortisol elevation as a result of public speaking in the first place (Blood, Blood, Frederick, Wertz, & Simpson, 1997). Compared to controls, people with a fear of public speaking experience increased stress, in the form of elevated heart rate, even when speaking to a simulated audience of "virtual" people (Slater, Pertaub, Barker, & Clark, 2006).

Affectionate Communication

The human species did not evolve—as some other primates did—to live a solitary life. On the contrary, *Homo sapiens* is perhaps the most social of the social primates, so it is unsurprising that the communication of affection plays an unparalleled role in the formation and maintenance of satisfying interpersonal relationships (see, e.g., Denes, 2012; Horan, 2012; Mansson & Booth-Butterfield, 2011). Beyond its relational functions, however, affectionate behavior has robust physiological effects, as research programs in both communication (Floyd, 2006a) and health psychology (Grewen, Girdler, Amico, & Light, 2005) have explicated. Much of Floyd's work has tested a fundamental tenet of affection exchange theory (AET; Floyd, 2002; 2006a), that expressing and receiving affection activates neuroendocrine responses that can mobilize the body's stress reaction. These effects have also been shown to operate separately for expressed and received affection (Floyd et al., 2005). Three patterns, in particular, have been identified: affectionate communication aids stress regulation; affectionate behavior acts as a stress buffer; and engaging in affectionate behavior accelerates physiological recovery from elevated stress. Consequent-

ly, increasing affection has also been shown to provide health benefits.

Affectionate communication aids stress regulation.

Floyd (2006b) hypothesized that affectionate communication aids in the regulation of stress. If so, then the amount of affectionate communication individuals express and receive on a regular basis should be associated with physiological markers of stress. One robust and commonly measured marker is diurnal (i.e., 24-hour) variation in cortisol. Strong diurnal variation in cortisol reflects a healthy ability of the hypothalamic-pituitary-adrenal axis to regulate the body's stress response, whereas attenuated variation reflects dysregulation (see, e.g., Giese-Davis, Sephton, Abercrombie, Durán, & Spiegel, 2004). After measuring self-assessed affectionate communication from 20 healthy adults, Floyd (2006b) collected saliva samples at four points over the course of a normal working day: upon awakening, at noon, in the late afternoon, and at bedtime. As hypothesized, affectionate communication showed a strong ($r = .56$) relationship to diurnal variation in cortisol. In an extension of the Floyd (2006b) study, Floyd and Riforgiate (2008) recruited 20 healthy adult participants and their spouses to provide reports of their verbal, nonverbal, and supportive affectionate communication. Participants also provided saliva samples for diurnal assessment of cortisol and dehydroepiandrosterone-sulfate (DHEA-S), a pro-hormone related to the stress response. As expected, spouses' reports of affectionate communication predicted participants' waking cortisol, diurnal change in cortisol, and cortisol: DHEA-S ratio (perhaps a more reliable marker of stress than the absolute level of either chemical alone; Cruess et al., 1999).

Floyd, Hesse, and Haynes (2007) showed that, among healthy adults, self-reported levels of affectionate communication are negatively related to resting blood pressure and blood glucose, the latter being a risk factor for diabetes. Among women, Light, Grewen, and Amico (2005) also found that the self-reported frequency of hugs from their husbands or partners predicted lower resting blood pressure and higher levels of circulating oxytocin. Similarly, in a study of mid-aged women (mean age = 47.6 years), Burleson, Trevathan, and Todd (2007) found using multilevel modeling that sharing physical affection with

a partner on one day predicted significantly lower stress the following day.

Affectionate behavior acts as a stress buffer.

The results of the aforementioned studies suggest a regulatory role for affection in the management of stress, but various experiments have also demonstrated that exchanging affectionate communication—either in general or immediately preceding a stressful event—attenuates physiological reactivity (i.e., serves as a buffer) to the stressor. For example, Floyd, Mikkelsen, Tafoya et al. (2007a) measured participants' assessments of the degree of affection present in their closest relationships. The researchers found that participants' reports of verbal and supportive affectionate communication were inversely related not only to their resting heart rate but also to their cortisol reactivity to laboratory stressors. The greater the level of affection participants reported having in their lives, the less their cortisol was elevated in response to the stressful activities.

Grewen, Anderson, Girdler, and Light (2003) found that adults who engaged in ten minutes of handholding and a 20-second hug with their romantic partner prior to a public speaking stressor demonstrated lower elevations in heart rate and systolic and diastolic blood pressure compared to a no-contact control group. Similarly, Ditzen et al. (2007) discovered that women with affectionate touching by a married or cohabitating partner before a laboratory induced stressor exhibited significantly lower cortisol and heart rate responses to the stress test.

Floyd, Pauley, and Hesse (2010) identified a possible mechanism for the buffering effect of affectionate communication in the pituitary hormone oxytocin. After measuring 100 adults' affectionate communication via diary for one week, the researchers exposed the adults to laboratory stressors similar to those used by Floyd, Mikkelsen, Tafoya et al. (2007a). The more affection participants had received in the week prior to the study, the greater their oxytocin elevation in response to the stressors. This finding is clinically significant because of oxytocin's parasympathetic effects and may possibly account for the attenuated cardiovascular responses identified by Grewen et al. (2003).

Affectionate communication accelerates recovery from elevated stress.

One published experiment has shown that when individuals are in a state of elevated distress, engaging in an act of affectionate communication can accelerate their physiological recovery. After exposing participants to standard laboratory stressors, Floyd, Mikkelson, Tafoya et al. (2007b) randomly assigned participants either to an experimental control or one of two control groups. Those in the experimental group wrote a letter for 20 minutes to a loved one in which they expressed their feelings of affection for that person. They were instructed to “tell this person why you love and care about him or her.” Participants in the first control group were instructed to think for 20 minutes about a loved one—and about why they loved and cared about that person—but not to put their feelings or thoughts into words. Those in the second control group sat quietly for 20 minutes.

The researchers monitored participants’ recovery from elevated cortisol in all three groups and found that participants in the experimental group returned to baseline cortisol levels significantly faster than did those in either control group.

Increasing affection provides health benefits.

If affectionate communication aids in regulating, buffering, and recovering from stress, then the potential exists that increasing affectionate behavior within existing supportive relationships is associated with health benefits. To date, a few experiments have demonstrated such benefits. In a four-week study, for instance, Holt-Lunstad, Birmingham, and Light (2008) taught spouses a “warm touch” support enhancement intervention and found that the participants experienced increased oxytocin levels and reduced levels of α -amylase and systolic blood pressure compared to spouses in a behavior monitoring control group.

Two other experiments have focused on blood lipids as a health outcome. Floyd, Mikkelson, Hesse, and Pauley (2007) assigned participants either to an experimental group in which they were asked to write about their affection for significant people in their lives for 20 minutes on three separate days or a control group where they were asked to write about innocuous topics. Total cholesterol was assessed at the beginning and end of the trial. Par-

ticipants who wrote about their affection had significant declines in their total cholesterol levels compared to the control group, which experienced an increase or no change at all in cholesterol levels. In a separate study, Floyd et al. (2009) assigned fifty-two healthy adults in married or cohabitating relationships either to an experimental group in which they were asked to increase the frequency of romantic kissing with their partner or to a control group. After a six-week trial, experimental participants experienced lower serum cholesterol, whereas no change in lipids was observed for control participants.

Continuing empirical work addresses the genetic and neurological substrates of affectionate communication. Using electroencephalography to examine neurological activity, Lewis, Heisel, Reinhart, and Tian (2011) discovered that high-affection communicators display greater relative electrical activity in the left anterior cortex versus the right anterior cortex, whereas the same asymmetry is not evident among low-affection communicators. This is noteworthy insofar as the left and right prefrontal cortices mediate tendencies for approach and avoidance. More recently, Floyd and Denes (2013) found that trait affection is higher for carriers of the GG allele (genetic form) on the oxytocin receptor gene rs53576 than for carriers of the AG or AA alleles, and that type of allele is more influential for those low in attachment security than for those high in attachment security. Theirs was the first study to document a genetic association with the tendency for affectionate communication. As studies similar to these are conducted, they will begin to shed light on the question of innateness—that is, how acquired or inborn is an individual’s level of affectionate communication?

Conflict

Whereas affectionate behavior buffers physiological reactions to stressors, conflict is frequently associated with dysregulated physiological stress responses. Insofar as conflict represents a struggle over resources, this is notable from an evolutionary perspective. When individuals engage in conflict, they risk losing resources and further risk retaliatory aggression from the other party. On the other hand, they stand to gain both resources and social capital if they succeed in the conflict. Evolutionarily, therefore, the presence of conflict is not necessar-

ily problematic; what would be problematic is a compromised ability to manage and recover from conflict.

Conflict can occur in any relationship characterized by interdependence, but the majority of research examining the physiological experience of conflict has focused on marital conflict. Two principal findings have been that a) various qualities of marital conflict affect spouses' physiological health; and b) spouses' physiological reactivity to their marital conflict predicts their relational quality and stability. Research demonstrating each effect is summarized in this section.

Quality of marital conflict affects physiological health.

A robust empirical literature documents associations between marital conflict and physiological health outcomes. Importantly, it is not the *presence* of conflict, *per se*, but the *quality* of conflict that is influential—individuals benefit physiologically not when they argue less frequently but when they argue with less hostility and greater positivity, according to research.

The work of Kiecolt-Glaser and colleagues has been foundational in establishing the relationships between the qualities of relational conflict and physiological health. In one experiment, Kiecolt-Glaser et al. (1993) reported that hostility during a marital conflict episode predicted down-regulation of the immune system. Specifically, hostility and negative behavior during spouses' conflict conversations predicted higher antibody titers to latent Epstein-Barr virus and greater decrements in natural killer cell lysis, blastogenic response to two mitogens, and proliferative response to a monoclonal antibody to the T3 receptor, all of which indicate detriments in immune system function. In separate analyses of the same couple interactions, Malarkey, Kiecolt-Glaser, Pearl, and Glaser (1994) found that hostility also predicted increases in epinephrine, norepinephrine, the stress hormone ACTH, and growth hormone, and decreases in the hormone prolactin. Additional research has found that variables such as spousal support satisfaction (Heffner, Kiecolt-Glaser, Loving, Glaser, & Malarkey, 2004) and spouses' relative power (Loving, Heffner, Kiecolt-Glaser, Glaser, & Malarkey, 2004) moderate the effects of hostility on physiological reactivity. Moreover, both Fehm-Wolfsdorf, Groth, Kaiser, and Hahweg (1999) and Kiecolt-Glaser et

al. (1996) reported higher physiological reactivity to conflict among women than among men.

Hostility in marital conflict has also been found to suppress the rate at which the body heals. Kiecolt-Glaser et al. (2005) administered blister wounds to 24 healthy married couples and assessed the rate of healing during two admissions to a hospital research unit. During the second admission, the couples discussed a marital disagreement that was coded for hostility (a composite of psychological abuse, distressing attributions, and critical or hostile behavior). The researchers found that couples who demonstrated high hostility healed at 60% the rate of the low-hostility couples.

Whereas hostile behaviors during marital conflict exacerbate negative physiological outcomes, positive behaviors enhance favorable physiological outcomes. For instance, Robles, Shaffer, Malarkey, and Kiecolt-Glaser (2006) examined conflict episodes of 90 newlywed couples using the Marital Interaction Coding System. Husbands' positive behavior—a composite of supportive communication, humor, and problem-solving behavior—predicted decreases in their wives' cortisol and ACTH during the conflict. In addition, supportiveness during highly negative conflict conversations led to steeper cortisol and ACTH decreases in wives. Robles et al. opined that the latter finding suggests that “constructive engaging in discussions promotes adaptive physiological responses to interpersonal conflict” (p. 305), a conclusion that is certainly consistent with Gottman's principle that conflict behavior favoring positivity by a substantial ratio is protective (1993).

Other research has examined which behaviors, specifically, lead to physiologically positive outcomes. Graham et al. (2009) found that spouses who used more cognitive processing words—those related to causal reasoning (e.g., because, why, hence), thinking (e.g., ought, should), and insight (e.g., know, realize, understand)—had significantly smaller increases in two cytokines, interleukin-6 and tumor necrosis factor- α , both of which are associated with inflammation. Some research has even tested interventional models. For example, receiving a dose of oxytocin intranasally prior to a conflict conversation has been shown to significantly increase spouses' positive communication behavior, and assuage cortisol increases, as compared to a placebo (Ditzen et al., 2009).

Physiological responses to marital conflict affect relationships.

Gottman's research has demonstrated that spouses' physiological reactions to marital conflict significantly predict the quality and stability of their marriages (Levenson, Carstensen, & Gottman, 1994). Although communication scholars are frequently familiar with Gottman's "four horsemen" and his ratio model of 5 positive behaviors for every 1 negative behavior in satisfied couples, many are unaware that Gottman's seminal research has supported two complementary physiological models for predicting marital distress and dysfunction. As Gottman and Levenson (1999) described, the first model—the *baseline arousal model*—provides that spouses' physiological arousal before beginning (i.e., while anticipating) a conflict conversation predicts declines in marital satisfaction and progress toward marital dissolution over time. Supporting the baseline arousal model, Levenson and Gottman (1985) found that higher autonomic arousal at the beginning of a conflict conversation predicted larger declines in marital satisfaction for 19 couples three years later. The second model—the *interaction arousal model*—claims that spouses' physiological arousal during a conflict conversation predicts marital dysfunction. In support of the latter model, Gottman and Levenson (1992) found in a study with 73 couples that wives' greater autonomic arousal (measured in this experiment as heart rate and finger-pulse amplitude) during marital conflict was associated with marital dissolution four years later.

In a summary and empirical comparison of the models, Gottman and Levenson (1999) noted that the baseline model suggests that spouses' physiological responses tap an expectation that their marital interaction will be aversive, whereas the interaction model suggests that spouses' responses to actual aversive events in the conversation are potentially predictive of later marital distress. Using different data from the 1985 and 1992 investigations, Gottman and Levenson (1999) found that the physiological models are equally capable of predicting relationship dissolution.

Additional support for the interaction model was later provided by Kiecolt-Glaser, Bane, Glaser, and Malarkey (2003). The researchers observed 90 couples take part in a conflict interaction during their first year of marriage and then assessed marital status (whether married or divorced) and marital quality (for those couples still mar-

ried) for all 90 couples ten years later. Among couples still married, current marital distress was predicted by higher levels of ACTH and norepinephrine during the Time 1 conflict interaction. Moreover, divorce during the ten-year period was predicted by higher levels of epinephrine during the Time 1 conflict interaction. Stated differently, the more physiologically aroused couples were during their conflict in their first year of marriage, the more likely they were to divorce within ten years or to be distressed if they were still married ten years later.

Divorce may also affect how adults perceive their own communication, which can influence their physiological responses to their communication patterns. In a study of parent-adolescent pairs asked to discuss something stressful related to the parents' relationship, Afifi, Granger, Denes, Joseph, and Aldeis (2010) found that when divorced and non-divorced parents had a more strained relationship, they experienced an increase in α -amylase after the conversation. When the parents had a less strained relationship, those who were still married experienced no change in their α -amylase. Parents who were divorced and had a less strained relationship experienced a more dramatic increase in their α -amylase immediately after the conversation, however. A similar trend occurred for the inappropriateness of the disclosures, with more inappropriate disclosures having a greater effect on divorced parents' α -amylase. Afifi, McManus, Hutchinson, and Baker (2007) speculated that divorced parents with a positive relationship may feel guilty when they speak badly about the other parent, which could be stress inducing. Parents with a strained relationship may also become desensitized to their conflict over time, making them less stressed and less cognizant of their communication with their child. If this mindlessness ensues, it could perpetuate even more inappropriate disclosures.

Social Support

Social support—the behavioral provision of encouragement, assistance, guidance, and companionship—is unquestionably adaptive for a social species such as humans. The receipt of social support not only aids individuals in dealing with acute stressors but also implies and reinforces the existence of reliable support bonds that would be expected to have both instrumental and psychosocial benefits for physical health.

Social support has health-protective effects.

A long history of studies has illuminated the protective effects of social support, both generally and specifically. Compelling evidence that having generally positive social relationships has health-protective effects comes from Cohen, Doyle, Turner, Alper, and Skoner (2003), who measured sociability levels (a composite of positive relationships with others, agreeableness, and extraversion) of 334 healthy adults. The adults were then exposed intranasally to one of two types of rhinovirus, quarantined for five days, and assessed to determine whether they developed a cold. Participants were considered to have developed a cold if the presence of an infectious agent was established and a threshold for respiratory symptoms (congestion, sneezing, sore throat, etc.) was met. Cohen et al. determined that, irrespective of age, preexisting antibodies, rhinovirus type, or other control variables, there was a negative linear relationship between sociability and susceptibility to the cold: the more positive one's social relationships, the lower the likelihood of contracting a cold (see also Cohen, Doyle, Skoner, Rabin, & Gwaltney, 1997; Cohen, Tyrrell, & Smith, 1991).

It was Cohen, too, who first advanced a stress-buffering hypothesis for social support, that "psychosocial stress will have deleterious effects on the health and well-being of those with little or no social support, while these effects will be lessened or eliminated for those with stronger support systems" (Cohen & McKay, 1984, p. 253). Multiple studies have demonstrated this hypothesized effect on physiological health outcomes. In an early study, Kamarck, Manuck, and Jennings (1990) found that the presence of a supportive friend reduced blood pressure reactivity to an acute laboratory stressor (a mental math exercise). Gerin, Pieper, Levy, and Pickering (1992) later found that participants' cardiovascular reactivity to a controversial discussion task was inhibited by the presence of a confederate who defended their position, as compared to a confederate who sat quietly.

The stress-buffering effect has also been demonstrated with endocrine and immune outcomes. Receiving support from a best friend during preparation for a public speaking stressor significantly reduced cortisol responses to the stressor (compared to no support; Heinrichs, Baumgartner, Kirschbaum, & Ehlert, 2003). Heinrichs et al. further found that the buffering effect of social support was en-

hanced by intranasal administration of oxytocin, such that those who received both social support and oxytocin experienced the least cortisol reactivity. Relatedly, Moynihan and colleagues (2004) found in a study of elderly nursing home residents that social support negatively predicted post-vaccine titers to the Panama strain of influenza, indicating a healthier immune response to the stressor of the vaccination for those with greater social support (see also Pressman et al., 2005). A meta-analysis of 22 experiments published between 1967 and 1998 found that the average effect size (*d*) of manipulated social support on reactivity to laboratory stressors was 0.61 for heart rate and systolic blood pressure, 0.51 for diastolic blood pressure, 0.25 for skin conductance, and 0.83 for cortisol (Thorsteinsson & James, 1999).

In addition to buffering individual reactions to stressors, receiving social support reduces the risk of complications during pregnancy (Elsenbruch et al., 2007), long-term chronic pain from rheumatoid arthritis (Evers, Kraaijmaat, Greenen, Jacobs, & Bijlsma, 2003), cognitive decline among healthy older adults (Seeman, Lusignolo, Albert, & Berkman, 2001), and recurrences of lesions from genital herpes simplex virus (VanderPlate, Aral, & Magder, 1988).

Social support can aid stress recovery and healing.

In addition to attenuating reactions to stressors, some investigations show that receiving social support also accelerates the body's ability to return to physiological baseline after exposure to stressors, as well as to heal from injury. Heffner et al. (2004), for example, found that wives' social support received from their husbands increased their ability to recover from stress after a conflict episode. They also found that newlywed wives and husbands had lower blood pressure after conflict when there were high levels of spousal support satisfaction. Roy, Steptoe, and Kirschbaum (1998) also found in their study of 90 male firefighters that social support predicted faster cardiovascular recovery from stressors.

Several studies have illustrated a relationship between social support and healing. Social support received by post-surgical cardiac patients predicts their speed of recovery and consumption of pain medication (Kulik & Mahler, 1989), emotional well-being, functional disruption, and angina (King, Reis, Porter, & Norsen, 1993),

and distress (Fontana, Kerns, Rosenberg, & Colonese, 1989) following surgery. Neuling and Winefield (1988) similarly showed that, for breast cancer patients, post-surgical anxiety and depression are negatively related to their satisfaction with the social support received from both family members and their surgeons. Research has also shown that social support in marriage accelerates wound healing, whereas exposure to chronic, daily conflict can slow healing rates by 40% (Kiecolt-Glaser et al., 2005).

Social support can increase physiological stress.

Paradoxically, receiving socially supportive communication from others does not always ameliorate stress or enhance well-being. Indeed, under some circumstances, it can elevate stress and contribute to negative health outcomes for patients and/or others. One reason why is that talking too much about one's own stressors can result in stress contagion, whereby an individual's distress spills over onto others (Afifi et al., 2007). Moreover, partners' stresses and coping mutually influence one another (e.g., Coyne & Smith, 1991). Specifically, verbally ruminating about stress, or "extensively discussing and revisiting problems, speculating about problems, and focusing on negative feelings" (Rose, 2002, p. 1830), is associated with depressive symptoms, anxiety, and heightened physiological responses to stressors (Afifi et al., 2007; Byrd-Craven, Geary, Rose, & Ponzi, 2008; Rose, Carlson, & Waller, 2007). In a study of problem-solving discussions among 88 female same-sex friends, Byrd-Craven et al. (2010) found that co-rumination was associated with increased cortisol levels after the problem-solving task. Negative affect predicted increases in cortisol and α -amylase after the task. The authors opine that future research should examine the role of dual activation models or situations in which α -amylase and cortisol have an additive effect and can adversely influence individual health.

Emotional Communication

Emotion is a drive force that motivates adaptive responses to changes in environmental conditions (see Ekman, 1992). Fear motivates people either to avoid or to neutralize threats; anger motivates retribution for losses so as to avoid future losses; embarrassment and

guilt dissuade inappropriate behavior; joy encourages social bonding; sadness elicits aid from others. Insofar as the experience and the expression of emotion are intimately linked, it is unsurprising that the expression of emotion—as opposed merely to the experience of emotion—is associated with multiple physiological effects. Much of Fredrickson's work, for example, demonstrates the stress-relieving physiological effects of positive emotion (e.g., Fredrickson, 2001; Fredrickson & Joiner, 2002; Fredrickson & Levenson, 1998). Two effects, in particular, that have received much empirical attention are the causal connection between expression and experience and the associations between decoding impairment and health.

Emotional expression induces emotional experience.

Although the physiological experience of emotion provokes emotional expression, experimental evidence also indicates a causal relationship between expressing emotion and experiencing it. According to the facial feedback hypothesis (Buck, 1980), adopting the facial expression of an emotion induces physiological responses consistent with that emotion. As Buck explained, the hypothesis can be traced to James's claim that peripheral bodily changes follow from perceptual and behavioral reactions to an event, including skeletal muscle reactions.

In one experiment, Levenson, Ekman, and Friesen (1990) had participants pose facial expressions for anger, disgust, fear, happiness, sadness, and surprise while their skin temperature, heart rate, and skin conductance were monitored. Analyses of group-level data found that poses of anger, fear, and sadness produced heart rate accelerations larger than for poses of happiness, disgust, and surprise. Fear and disgust increased skin conductance, and anger increased skin temperature. Analyses of individual-level data found that when participants produced facial expressions that most closely resembled the associated emotion, the physiological differences between the emotions were the most pronounced, and their self-reports of the associated emotion were the most prevalent. Levenson et al. further found that their results were valid for both sexes and were not limited to actors or to scientists who study the face. Zuckerman, Klorman, Larrance, and Spiegel (1981) had earlier found that inducing participants to mimic facial expressions of emotion seen on a film

increased the participants' physiological arousal as measured by heart rate, skin conductance, and blood pressure.

A later experiment by Hess, Kappas, McHugo, Lanzetta, and Kleck (1992) assigned female undergraduates to complete three tasks: 1) feel four emotions (happiness, anger, sadness, peacefulness) without expressing them; 2) express those emotions without feeling them; and, 3) feel and express those emotions. During each task, participants pressed a button to indicate when they had reached the indicated state. The researchers measured the latency from emotion cue to button press, as well as participants' heart rate and skin conductance for 15 seconds before and after each button press. Consistent with the facial feedback hypothesis, expressing emotion without feeling it resulted in heart rate changes equal to those produced by feeling the emotion, although there was no effect for skin conductance. Moreover, participants had a shorter self-generation latency in the feel-and-express condition than in the feel-only condition, suggesting that expressing facilitates feeling (as the facial feedback hypothesis claims).

Consistent with the facial feedback hypothesis, some studies have also shown that the physiological characteristics of an experimental manipulation influence people's self-reported emotional experiences. Strack, Martin, and Stepper (1988) had participants hold a pen in their mouths in a way that contracted either the zygomaticus major or risorius muscles (facilitating a smile) or the orbicularis oris muscle (inhibiting a smile). Those in the former group reported more positive affect than those in the latter group in response to cartoons. Larsen, Kasimatis, and Frey (1992) applied the same principle to testing unpleasant affect, finding that inducing contraction of the corrugator supercilii muscle resulted in significantly more sadness in response to aversive photographs than the lack of contraction.

Decoding impairment influences physiology.

The ability to decode emotional expressions accurately is unquestionably adaptive for survival within a social species. For example, conspecifics' expressions of fear alert one to threats, whereas expressions of joy advertise social opportunities and expressions of disgust imply the presence of potentially toxic foods or fumes. Those unable to interpret such cues would be distinctly

disadvantaged with respect to survival, all else being equal. It is therefore not surprising that alexithymia, a syndrome marked by the inability to decode expressions of emotion (Taylor & Bagby, 2013), is linked to compromised health and aberrant patterns of physiological stress management.

Research has found alexithymia to be associated with numerous somatic pathologies, including dermatological conditions such as psoriasis, alopecia areata, atopic dermatitis, and chronic urticaria (Willemsen, Roseeuw, & Vanderlinden, 2008), near-fatal asthma (Serrano et al., 2006), pain intensity among muscular dystrophy patients (Hosoi et al., 2010), and glycemic control among child diabetics (Housiaux, Luminet, Van Broeck, & Dorchy, 2010). Alexithymia is also associated with suppression of the immune system. Dewaraja et al. (1997) found that alexithymic men had decreased counts of particular immune system cells (for the natural killer subset: CD57-CD16+ cells and killer effective T cell CD8+CD11a+ cells) compared to non-alexithymics. Similarly, Todarello et al. (1994, 1997) reported lower counts of nearly all immune cell subsets in alexithymic women than in non-alexithymic women (see also Corcos et al., 2004). In a recent study with middle-aged men in Finland, Tolmunen, Lehto, Heliste, Kurl, and Kauhanen (2010) found that the risk of cardiovascular mortality was increased by 1.2% for each 1-point increase on the Toronto Alexithymia Scale, the most frequently used measure of alexithymia.

Alexithymia is also associated with abnormal patterns of physiological functioning, both at rest and in response to stimuli. At rest, physiological activity is heightened in people with alexithymia. Compared to a reference population, alexithymics exhibit higher cardiac output (Paciac, Feuerstein, & Spiegel, 1985) and higher electrodermal activity (Friedlander, Lumley, Farchione, & Doyal, 1997), consistent with being in a state of heightened alert facilitated by the inability to interpret social signals.

On the contrary, however, experimental research generally shows that when presented with what would normally be emotionally evocative stimuli, individuals with alexithymia exhibit blunted physiological responses, which is again consistent with their relative inability to interpret those cues. Wehmer, Brejnak, Lumley, and Stettner (1995), for instance, found that people with alexithymia had smaller heart rate increases and fewer electrodermal responses to emotionally evocative slides compared to a

control sample. Roedema and Simons (1999) demonstrated the same effect for skin conductance responses. In an fMRI study, Hesse et al. (2013) also found that exposure to emotionally positive images produced lower hemodynamic activity for alexithymic than non-alexithymic participants in the left inferior temporal/temporal occipital fusiform, the right lingual/parahippocampal gyrus, the right hippocampus, the left middle occipital/middle temporal gyrus, the left hippocampus, and the right temporal pole/superior temporal gyrus. Similarly, Berthoz et al. (2002) found that alexithymic men had less activation in the left mediofrontal-paracingulate cortex when viewing emotionally negative photographs—although more activation in the anterior cingulate, mediofrontal cortex, and middle frontal gyrus when viewing emotionally positive photographs—compared to non-alexithymic men.

Aggression

From an evolutionary standpoint, a tendency toward aggression is both an asset and a liability. In any species, aggressive behavior can establish dominance, defend territory, and help to ensure access to resources and mating opportunities. It is also costly in terms of energy consumption and the potential for injury and retaliatory aggression. Among humans, aggressive behavior is observed in forms that are socially sanctioned, such as during military combat, athletic competition, and self-defense. It also occurs in forms that are socially proscribed, as during the commission of a crime. In either case, injury and retaliation are evident risks, making a tendency toward aggressive behavior both advantageous and disadvantageous when compared to a tendency toward passivity.

Of all biological factors, testosterone is most commonly studied in terms of its relationship to aggression. Despite popular perceptions of testosterone-fueled aggression, the relationship between aggressive behavior and testosterone is complex. Some studies have found linear relationships between testosterone and aggressive behavior. Soler, Vinayak, and Quadagno (2000), for instance, found that men's testosterone levels predicted both their verbal aggression and physical aggressive behavior toward their domestic partners. Among men, aggression is also related to finger length ratio (2D:4D), a sexually dimorphic trait that reflects prenatal testosterone levels

(Bailey & Hurd, 2005).

Some experiments have examined the effects on behavior of administering testosterone to participants. For instance, O'Connor, Archer, and Wu (2004) administered either testosterone or placebo to 38 healthy men and found that testosterone did not increase aggressive behavior but did significantly increase hostility and anger relative to baseline. Similarly, in an fMRI study of women, sublingual administration of testosterone (versus placebo) increased neural activation in response to angry faces (versus happy faces) in areas known to be involved in reactive aggression, such as the amygdala and hypothalamus (Hermans, Ramsey, & van Honk, 2008).

Even symbols of aggression can elevate testosterone. Klimesmith, Kasser, and McAndrew (2006) had male undergraduates interact either with a gun or a children's toy gun for 15 minutes. The participants then added as much hot sauce as they wanted to a cup of water they believed another participant would have to drink. Compared to men who interacted with the toy, those who interacted with the gun had significantly greater increases in testosterone, and also added significant more hot sauce to the water.

Although those studies demonstrate linear relationships, there are at least three reasons to conclude that the actual relationship is more complex. For one, not all studies have found a significant relationship. Coccaro, Beresford, Minar, Kaskow, and Geraciotti (2007) found no relationship among men after measuring testosterone from cerebrospinal fluid. Carré, Putnam, and McCormick (2009) likewise found no relationship between baseline testosterone and aggressive behavior for either women or men, although they did find that changes in testosterone concentrations over time predicted aggressive responses to a competitive computer activity. Among early adolescent boys (ages 6 to 12 years), Schaal, Tremblay, Sousignan, and Susman (1996) even found that a history of high physical aggression was related to lower testosterone, rather than higher.

Second, average effect sizes are small. In a 2001 meta-analysis, Book, Starzyk, and Quinsey found a mean weighted correlation of only 0.14 between testosterone and aggression, although a published reanalysis of the data reported an even lower mean weighted correlation (0.08; Archer, Graham-Kevan, & Davies, 2005). A more recent meta-analysis of the relationship between aggres-

sion and digit ratio (2D:4D) found only a modest relationship (0.06) and only for men (Hönekopp & Watson, in press).

Finally, research suggests that the influence of testosterone on aggressive behavior is moderated by environmental and physiological factors. In an experiment with undergraduate men, for instance, Carré, Gilchrist, Morrissey, and McCormick (2010) found that situational and motivational factors influence the relationship between testosterone and aggressive behavior. Participants played a computer-based activity in which they press a button to earn points, which they can later exchange for money. A fictitious opponent steals points from them, and they can steal points from the opponent. The researchers crossed two experimental variables to produce four conditions. The first variable was whether aggressive behavior was rewarded (participants keep their stolen points) or costly (stolen points are not kept and prevent future points from being earned); the second was whether or not aggression was provoked by previous stealing from the opponent. Carré et al. found a correlation between testosterone and aggressive behavior only in the provoked/rewarded condition, in which participants are provoked during the task and told they get to keep any points they steal.

Similarly, Popma et al. (2007) found that the relationship between testosterone and aggression is moderated by cortisol. In a study of adolescent boys referred to a delinquency diversion program, the authors found a significant positive relationship between aggression and testosterone among participants with low cortisol levels, but no significant aggression-testosterone relationship among participants with high cortisol levels.

To be certain, communication apprehension, affectionate communication, conflict, social support, emotional communication, and aggression are not the only communicative behaviors whose biological antecedents, consequences, and correlates have been identified. These behaviors represent a sample, not an exhaustive list, of the links between biology and communication that have been identified by research primarily in other disciplines. As the next section contends, the communication discipline's ignorance of biology and evolution as causal factors has led the field to a tenuous theoretic position.

Biology, We Ignore Thee at Our Peril

As evidenced by the previous section, research on the biological and evolutionary antecedents, consequences, and correlates of communication behavior is thriving. With few exceptions, however, it is not thriving within the discipline of communication. Not only is most of the research not being conducted by communication scholars; most of the research remains entirely unknown to communication scholars. It is not cited in communication textbooks or in articles published in communication journals. This is true despite the fact that all of the research reviewed in the previous section focuses explicitly on communication behavior.

Ignorance of the bio-evolutionary roots of humans and human behavior—whether accidental or intentional—does more than keep the field unaware of other disciplines' discoveries. It also maintains the communication field in a tenuous theoretic position, exemplified by at least three problems described in this section.

Humans Are Animals Too

The first problem is that communication theory is needlessly anthropocentric, meaning that theories generate human-specific explanations for behaviors that are not unique to humans. Many behaviors that communication researchers study are truly human-specific. No other species uses Twitter, engages in gossip, manages medical information, or ponders academically the significance of Snooki (Goldthwaite Young & Esralew, 2011). By contrast, many other communication behaviors are observed across species, including deception; power and dominance; conflict; affection and intimacy; expressions of fear, anger, surprise, jealousy, sorrow, and other emotions; caregiving; and instruction and modeling. With few exceptions, however, communication theories provide accounts for these behaviors that are unique to the social and cognitive realities of humans.

One example is the well-documented finding that women contribute more time and energy to childcare than men do, on average (see, e.g., Thompson & Walker, 1989). Why is that the case, however? Theories used in the communication field offer a variety of explanations. Gender socialization theories propose that children observe their mothers performing more of the childcare than their fa-

thers and thereby learn gender-appropriate family roles (e.g., Maccoby, 1990). Of course, such theories leave entirely unanswered the question of why the sex difference exists in the first place, however; they explain only how the difference is perpetuated intergenerationally. Feminist theories tend to attribute the sex difference in childcare labor to patriarchy and a long history of men's political subjugation of women (e.g., Calasanti & Bailey, 1991). Alberts, Tracy, and Trethewey (2011) recently offered an "integrated" theory proposing that the sex difference in childcare and other domestic labor is due to an interaction of social exchange and economy-of-gratitude factors.

Inconveniently for all of these theories, however, the sex difference in childcare effort is not unique to humans. Females contribute more time and energy to childcare than males do among a variety of species, including spiders (Rypstra, Wieg, Walker, & Persons, 2003), hummingbirds (Lack, 1968), rhinoceros (Hutchins & Kreger, 2006), and armadillos (Newman, 1913). Do an economy of gratitude or the political subjugation of females account for this difference among these species as well? If not, then it is worth asking why a social behavior that is not unique to humans requires a theoretic explanation that is.

Parenting Is More Than Teaching

A second problem is that similarity in communication behavior is frequently attributed to environmental causes even when heritability effects are probable. As communication researchers have examined the question of acquisition—how it is that people acquire their communication traits—they often have looked for and found similarity between the behaviors of parents and their children. For instance, Plax, Kearney, and Beatty (1985) found a positive relationship between children's assertiveness and their perceptions of their parents' assertiveness. Similarly, Hutchinson and Neulip (1993) found a positive relationship between parents and children's communication apprehension. When such similarity is identified, however, it is almost always attributed exclusively to the effects of social learning and modeling, even when heritability is an equally plausible explanation.

As one example, communication research has established that verbally aggressive parents tend to have verbally aggressive children. In one study, Martin and An-

derson (1997) examined the correlations between college students and their parents' scores on argumentativeness, assertiveness, and verbal aggressiveness. Although Martin and Anderson acknowledged the possibility of a genetic basis for communication traits in their literature review, their hypotheses were firmly rooted in Bandura's social cognitive theory. Further, of the 160 father-mother-child triads in their study, there was no differentiation noted between biological and non-biological (e.g., step-parent, adoptive parent) parental relationships, precluding even an examination of whether parent-child correlations are stronger in biological than non-biological relationships.

The problem with that approach—which is the rule, not the exception, in communication research—is that some communication traits, including aggressiveness, are partly heritable, meaning they are passed from parents to offspring genetically. A meta-analysis of studies comparing monozygotic and dizygotic twins reported that the communication trait of aggressiveness is 58% heritable (Beatty, Heisel, Hall, Levine, & La France, 2002). That means that studies such as Martin and Anderson's, which presume that children acquire their communication traits from their parents only through learning, are misappropriating substantial proportions of variance. Specifically, if a study recruits biological parent-child pairs in which the parents have raised the children, then environmental and genetic effects are confounded. If the researchers in that study identify a significant parent-child correlation in a communication trait and attribute it to the effect of modeling, they are automatically over-estimating the effect of modeling because they have not partialled out the heritability effect. This unfortunate problem is the result of the discipline's biological illiteracy.

They Don't Live Among Us

Finally, by focusing such exclusive attention on proximal causes for behavior, the communication discipline often treats entities such as media and culture as though they were independently living organisms. This would be innocuous except that attributing behavior to an entity such as media or culture—as though it lives independently—fuels the practice of blaming such entities for bad behavior. Criminal defense attorneys blame their clients' actions on news media, movie violence, or social pressures. The more that culture, media, and "society" are

conceptualized as living entities, the easier it becomes to impute bad behavior to them.

The problem with that conceptual approach is that media, culture, society, and related entities do not exist independently from humans. They are human creations. There is no media, no culture, and no society without humans deciding what those entities are, what they do, and how they should be maintained. Human decisions and actions can cause their fundamental nature to change and can even lead to the extinction of various cultures and media forms. Theoretically, therefore, it makes no more sense to blame a crime on media than on the car that drove the criminal to the scene. Both media and the car are impotent without the human inputs—thus, it is the humans providing those inputs who justly deserve the blame—yet by treating media (and culture, etc.) as a living entity, communication research encourages such irrational thinking. (Not all conceptualizations of media treat it as independent, of course—actor-network theories are one exception; see Latour, 2005.)

Communication may not be the only academic field to suffer from these three problems—being overly anthropocentric, ignoring heritability, and treating non-living entities as independent—but they can all be traced to a lack of awareness about the bio-evolutionary roots of human behavior. Thankfully, the discipline is not doomed to intellectual stagnation. As the final section details, moving forward requires a commitment to eschew the false dichotomy of “nature vs. nurture” and to embrace the reality that both nature and nurture are largely ineffectual in shaping communicative behavior on their own.

A Way Forward

As Celeste Condit opined in her National Communication Association Carroll C. Arnold Distinguished Lecture, “We can no longer afford the insularity of ignorance about the biological inputs to human beings” (Condit, 2006, p. 20). Eschewing that insularity—and avoiding the intellectual obscurity it eventually brings—does not require the communication discipline to abandon its focus on proximal causes to behavior. What it will take is a commitment to learning about the ultimate bio-evolutionary causes that *also* shape behavior. To do so, we must first reconsider the relationship between nature and nurture,

and then craft our questions in ways that make use of bio-evolutionary principles.

The End of Nature vs. Nurture

Toward that end, it is past time for the discipline to grow beyond the false dichotomy of *nature vs. nurture*. When it comes to human social behavior, the reality is that nature and nurture rarely exert truly independent influences. For instance, Soler et al. (2000) found that testosterone was significantly related ($\beta = .26$) to men and women’s self-reported verbal abuse against their romantic partners. That result leaves substantial variance unaccounted for, however, meaning that although testosterone is potentially a contributory factor, its influence is likely moderated by other factors that also account for variance in romantic partner verbal abuse, such as time in the relationship (Roberts, Auinger, & Klein, 2006), presence of both parents in the household while growing up, age at leaving secondary school, and adolescent substance abuse and delinquency (Magdol, Moffitt, Caspi, & Silva, 1998). At most, one could claim on the basis of Soler et al.’s finding that a high testosterone level predisposes individuals toward verbally aggressive behavior, not that it necessarily acts independently of individuals’ experiences or environments to cause such behavior.

In fact, many biological contributors to social behavior are known to interact with identified environmental factors. For instance, variants at the serotonin transporter (*SLC6A4*) and 2A receptor (*HTR2A*) genes predict cooperative behavior (Schroeder, McElreath, & Nettle, 2013)—but these genetic variants predict cooperative behavior differentially depending on whether or not the cooperation can be punished (a purely environmental variable). This result reflects the phenomenon of epigenetics, in which genetic expression—but not genetic structure—is altered by environmental influences (Holliday, 2006). Even in the case of Schroeder et al., however, the interaction between genetic variation and the presence of punishment does not account for 100 percent of the variance in cooperative behavior, meaning that other factors—biological, social, or both—are also operative.

Biological factors rarely, if ever, act in isolation to *cause* social behaviors, even if they serve to motivate or increase the likelihood of those behaviors under certain conditions. As Floyd (Floyd & Haynes, 2005), Sherry (2004), and

others have noted, the backlash in the communication discipline against the claim of *biological determinism*—the contention that genetic and physiological characteristics determine behavior—has been largely without merit, as no such claim is advanced in the fields of evolutionary psychology, behavioral psychiatry, or psychophysiology (see, e.g., Barkow, Cosmides, & Tooby, 1992). In contrast, however, the communication field has tended to theorize under an uncontested assumption of *environmental determinism* (see Meaney, 2001) implying that malleable environmental characteristics (parenting behaviors, media images, cultural messages) determine behavior.

There can be little question that those and other environmental characteristics influence behavior—but they cannot do so except in interaction with biological factors. The reason is that parenting behaviors, cultural traditions, media messages, and other such factors exert their influence via learning, and the capacity to learn is neurologically restrained. Only those individuals who have achieved sufficient neurological development and are free of necessary neurocognitive impairments are able to learn and to have their social behavior shaped by such factors. Given sufficient neurological impairments (e.g., Alzheimer's disease, Downs syndrome, autism) and/or deficient neurological development (i.e., insufficient age), enculturation, direct instruction, modeling, media effects, and other sources of environmental influence are impotent.

Moreover, environmental factors can have no effect on communicative behaviors unless individuals have the biological potential both to be influenced by the environmental factor and to enact the communicative behavior. Speaking, listening, gesturing, making and interpreting facial expressions, and virtually all other communication tasks require specific anatomical and physiological abilities to perform. Exposure to violence in video games predicts aggressive behavior, for instance (Bartholow, Sestir, & Davis, 2005), but probably not for people who are blind and therefore lack the biological potential to be exposed to the violence in the first place. Similarly, cultural norms for politeness may teach that smiling is appropriate during an introduction, but those norms are ineffectual for people with Möebius syndrome, a genetic form of facial paralysis that makes smiling impossible (Kumar, 1990).

Using the Bio-Evolutionary Perspective

Communication scholars of all stripes can use bio-evolutionary theory and methods to illuminate their research questions. To do so requires paying attention to bio-evolutionary causation, ruling out alternative hypotheses, considering context carefully, and collaborating with experts when appropriate. The advice given here reiterates that offered by Floyd (2004), Floyd and Afifi (2011), and Floyd and Haynes (2005).

Conceptualize questions as bio-evolutionary.

The first step in using the bio-evolutionary perspective is to identify where it fits. Doing so requires considering how a given communication behavior may ultimately enhance or inhibit survival and/or procreation. Some links between communication and survival/procreation are relatively straightforward, such as the link between flirting and (eventual) procreation or the link between aggression and survival. Others may be less apparent, but there nonetheless. Deception, for instance, may ultimately serve both survival and reproduction motives. The point is for researchers to look beyond proximal influences such as cultural norms, expectancies, and media messages, to consider how communication behaviors fit longer-term ultimate goals.

Conceptualizing questions as bio-evolutionary also involves considering the biological antecedents and/or consequences of a communication behavior. Floyd and Afifi (2011) claimed that all interpersonal communication acts are biological acts, insofar as they require anatomical and physiological interaction to enact. One might expand that claim to virtually all communication acts, given that either encoding or decoding requires the use of biological faculties. As a result, scholars can consider how the biological characteristics of a communication act may serve its ultimate evolutionary motives. For example, the emotion of surprise arouses the sympathetic nervous system and the facial expression of surprise typically includes wide eyes with dilated pupils (useful for taking in visual information) and an open mouth (useful for increasing pulmonary volume). All of these biological characteristics help the emotion of surprise to contribute ultimately to survival by allowing an organism to assess and react to a potential threat.

Craft hypothesis tests to rule out rival explanations.

Although controlling for alternative explanations is a basic precept of the scientific design, it is of particular importance for scholars studying communication from a bio-evolutionary perspective. The primary reason is that it is often possible to deduce the same prediction using both bio-evolutionary and socio-cultural theories. For example, Floyd and Morman (2001) predicted on the basis of the theory of natural selection that men show more affection to biological sons than to stepsons. Their hypothesis reflected the evolutionary perspective that biological sons contribute more than stepsons to their fathers' reproductive fitness, but such a prediction could also be based on the reasoning that men feel closer to biological sons due to their extended relationship history. To support a bio-evolutionary hypothesis, therefore, Floyd and Morman had to control for factors such as closeness and relationship history.

Importantly, controlling for a rival explanation does not mean that the rival explanation is invalid. In Floyd and Morman's study, for instance, men did feel closer to biological sons than stepsons and that difference did account for some variance in affectionate behavior. The point of controlling for that explanation is to determine whether the evolutionary hypothesis is still valid—in other words, whether the difference between biological sons and stepsons remains even after alternative sources of variance are controlled.

Controlling for socio-cultural explanations also does not provide unqualified evidence in support of bio-evolutionary hypotheses. It is always possible that additional factors left unaddressed are accounting for variance. Ruling out alternative explanations does strengthen one's claim, however, and is good scientific practice whether testing bio-evolutionary predictions or not.

Consider context carefully.

A great risk when applying evolutionary theory to human behavior is to oversimplify evolutionary influences by failing to consider the social or relational contexts in which they occur. As explained above, evolutionary motives often interact with the context, and ignoring that interaction produces flawed hypotheses. If we consider only natural selection pressures as a predictor of behavior,

for instance, we might predict that individuals give twice as many social and tangible resources to full-biological siblings than to half-siblings because the level of genetic relatedness is 50% in the former relationship but only 25% in the latter. Such a prediction is flawed, however, because although evolutionary pressures toward survival and reproduction are an influence on behavior, they are almost never the sole influence. To apply bio-evolutionary reasoning properly, researchers must consider the social and relational context in which the evolutionary pressures are manifested.

Collaborate when necessary.

Although communication scholars are well able to apply bio-evolutionary reasoning to their predictions, many are inhibited by a lack of training and resources when applying psychophysiological methods to those predictions. One need not become a biologist to use physiological research methods, but it is important to understand the physiology of the outcomes one wishes to measure and to have access to appropriate equipment for data collection and analysis. Safety issues for both participants and researchers are also necessary to consider, especially when collecting fluid samples. Researchers who are untrained or inexperienced in these methods can benefit from collaboration with more experienced scholars.

The communication discipline is primed for a renaissance of understanding by nurturing an appreciation for what nature does to social behavior. That renaissance is already well underway in the laboratories of Afifi, Beatty, Floyd, Heisel, Hesse, Lang, Sawyer, Weber—and dozens of others outside the communication field. The methods and insights of this approach can exponentially broaden the discipline's understanding of communicative behavior. It remains only to be seen whether the field will choose to embrace these advances or remain needlessly myopic in its approach.

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Acknowledgements

Note from the Editor: Dr. Tim Cole (DePaul University) and Dr. Tsai-Shan Shen (Eastern Michigan University) have served as blind reviewers for this article. Dr. William Evans (University of Alabama) has participated as Open Peer Reviewer. After the acceptance of the manuscript they have agreed to sign their review. A third peer reviewer prefers to remain blind. I would like to thank them all very much for their time and valuable help.

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